DISASTER MEDICINE:
CONVENTIONAL TERRORIST BOMBINGS

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ABSTRACT

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The incidence of terrorism is increasing throughout the world. Bombing is a common form of terrorism that frequently results in multiple victims who are injured or killed. Survivors of terrorist bombings that arrive at civilian emergency departments have injuries that are different from other forms of trauma. The medical personnel treating these victims often have only limited knowledge of blast physics, mechanisms and determinants or injury, and the epidemiology of bombing injuries. This paper will review these issues and make recommendations for the medical management of bombing victims.

TABLE OF CONTENTS

ACKNOWLEDGEMENT

LIST OF ILLUSTRATIONS

x

LIST OF TABLES

INTRODUCTION

Terrorism is an act designed to generate fear, intimidate or coerce, affect government conduct, or punish a specific target,[1] and to maim as many people as possible in order to gain news media attention.[2] One of terrorists' principal weapons has been the bomb; either left indoors in public places or placed in a vehicle.[3] Terrorist bombings have become a common occurrence throughout the industrialized world. From 1969 to 1983, 220 terrorist bombings worldwide killed 463 persons and injured 2894.[4] Over the next 10 years, the number of terrorist bombings or bombing attempts increased 400% in the United States: from 803 in 1984 to 3163 in 1993. The World Trade Center bombing in New York City in 1993 resulted in more than 1000 injuries, 6 deaths, and $510 million in damage.[5] In the 1995 Oklahoma City bombing, there were 168 persons killed, 83 hospitalized and 509 treated as outpatients.[5,6] Thus, civilian emergency departments in the United States must be prepared for the possibility of large numbers of bombing victims.

By nature, a terrorist bombing is an isolated event. United States federal, postal, and military facilities are considered targets for terrorist bombings.[6] The Oklahoma City Alfred P. Murrah Federal Building in 1995 and the Navy destroyer USS *Cole* in 2000 are well-publicized examples.

Blast trauma from bombing incidents is highly variable, depending on the physical environment (e.g., indoors or outdoors, structural characteristics of building or vehicle, proximity to reflecting surfaces), device and explosive properties, victim populations, and victim distance from the blast and position relative to the blast wave.[3,7]

Explosions may kill or maim a victim in several ways. The blast wave may cause internal damage in air-containing organs without any external signs of trauma. A blast may propel fragments into a casualty or may propel the victim to impact on stationary objects or structures. Terrorist bombs are usually small, often weighing only a few kilograms. This allows them to be hidden in areas where unsuspecting victims are likely to congregate. The biological damage caused by these small bombs is greatly increased when they are packed with small metal objects such as bolts and nails.[8,9] This tends to generate many casualties and multiply the publicity impact for the perpetrators.

Victims are most likely to be treated at the site or in nearby Emergency Departments (ED) that can be quickly reached by ground transportation. Most emergency departments are unprepared for the sudden influx of perhaps hundreds of bombing victims. Casualties arriving to the emergency rooms after a bombing often arrive with complicated injuries that do not fall

within the daily routine of the ED staff. To ensure proper planning and to be fully prepared to treat bombing casualties, medical personnel should be familiar with blast physics, mechanism and determinants of injury, and epidemiology of bombing injuries.

BLAST PHYSICS

In the 18th century, Pierre Jars first described the injurious effects of explosions: "la grande et prompte dilation d'air."[10] He and his contemporaries pointed out that the variation of pressure in air resulting from the explosion was the cause of injuries.

Conventional terrorist bombs contain hydrogen, oxygen, nitrogen and carbon compounds in various solid, slurry, liquid, or gas states,[11] and require a detonator to initiate the explosion. If a volatile fuel is added, more heat and destruction are produced.[8] Terrorist bombs are encased in metal or plastic containers and range in size from only 2-30 pounds for those delivered in a suitcase or parcel to 500 pounds or more placed within vehicles.

Conventional high-energy explosives such as trinitrotoluene (TNT) combust rapidly and generate an expanding sphere of hot dense gas (the blast) that can have a pressure of over 10×10^6 psi.[12] This pressure transmits itself radially into the surrounding medium creating a blast wave (or shock wave), which has three features: a positive phase, a negative phase, and the mass movement of wind (blast wind). The combination of weight and distance from the explosion produces a particular pressure-time history and the laws of physics governing blasts are described by certain mathematical equations. The destructive capacity of a blast is due to the force it exerts (blast loading), which is usually described as a force per unit area.[11]

The defining characteristic of a blast wave at any point in space is the variation in ambient pressure over time. (**Figure 1**) During the positive phase, the wave increases in pressure rapidly in relationship to the explosion and rises above the ambient air pressure (blast overpressure). The peak pressure and duration of the positive phase are a function of the size of the blast and the distance from the blast.[13] The biological response to the blast wave from conventional explosives is dependent predominantly upon the peak overpressure and the duration of the positive phase.[14]

Overpressure

Peak

Impulse

**Normal
Atmospheric
Pressure**

Time

Positive Phase　　　**Negative Phase**
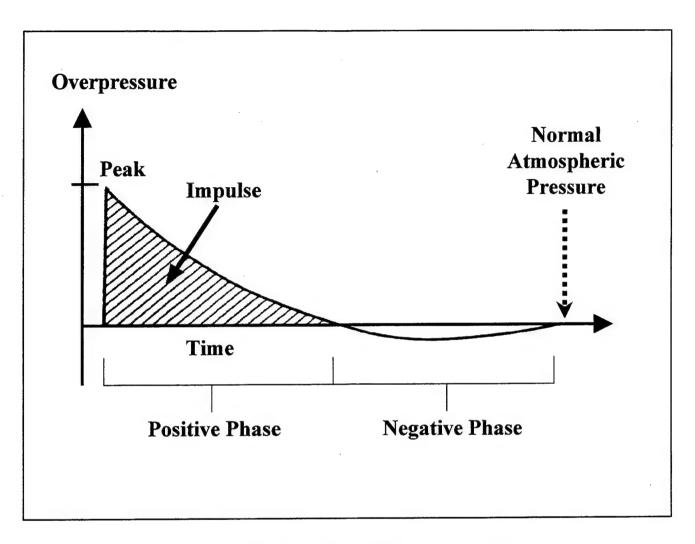
Figure 1: Pressure-Time History of an Air Blast

The ideal pressure-time history of an air blast in an undisturbed, free-field environment. The impulse is the integral of pressure over time. Modified from Stuhmiller JH, Phillips YY, Richmond DR: The physics and mechanisms of primary blast injury, in Bellamy RF, Zajtchuk R (editors): Conventional Warfare: Ballistic, Blast, and Burn Injuries. Textbook of Military Medicine Series, Office of the Surgeon General at TMM Publications, Washington, D.C., 1991.

Blast winds result from large volumes of air that are displaced by the expanding gases of the explosion.[8] The blast strength is the ratio of overpressure to the ambient atmospheric pressure. The term blast front is used to describe the leading edge of the blast wave. The blast wave propagates outward through the air at supersonic speed (velocities of 3000-8000 m/sec), but loses its pressure and velocity exponentially with distance from the source.[3,8,13] Because

water is incompressible, underwater wave propagation has a much greater speed and loses energy less quickly with distance.[13]

Because the speed of propagation increases with pressure, the negative phase moves more slowly than the positive phase of the blast front.[8] The negative phase can last 10 times longer than the positive phase and results in pressure below ambient air pressure. During the negative phase, the blast wind reverses back towards the direction of the explosion. This sucks debris into new areas.

Blast pressures are defined as static, dynamic, or reflected.[11] The static pressure is the air compression that is active in all directions due to the thermal motion of the gas. The dynamic pressure is the force associated with the movement of air particles at the leading edge of the shock wave. Blast waves that encounter a large, solid barrier (e.g., the wall of a building) in a perpendicular direction will result in a compression of molecules until they are so tightly packed that they push back in the direction of the incident wave. As the blast winds become stronger, the overpressure in the reflected region grows proportionately and can reflect off solid objects at 2 to 9 times the initial peak pressure.[8] Therefore, blast waves that will cause only modest injury in the open can be lethal if the victim is standing near a reflecting surface.[13] The "fireball" from the explosion may ignite clothing, surrounding objects or structures and cause burn and inhalation injuries.

Blast waves inside an enclosed structure (e.g., building, bus, foxholes, or armored vehicle) undergo repeated reflections from the interior surface and create a complex blast wave with three characteristics: (a) the incident blast wave, (b) a jumble of reflected waves, and (c) the static pressurization of the enclosure.[11,14] Indoor blasts can be associated with severe injuries because of the geometric increase in the pressure wave as it is reflected off walls, floors, and ceilings.[4] The intensity and duration of this pressure depends on the volume of the enclosed structure, and the degree of venting through the doors and windows.[14]

The blast loading on a human being is the amount of force exerted on the body and is affected by the body position in relation to the blast wave. If the maximum overpressure and duration of the positive incident overpressure remain constant and the presence of projectiles ignored, the incidence of physical injury and mortality will be greater when a person is oriented with the long axis of their body perpendicular (oriented side-on) and near a reflecting surface. Those not near a reflecting surface but perpendicular to the blast winds will have a lower incidence of physical injury and mortality. The lowest level of injury and mortality from the blast wave will be among victims whose long axis of the body is parallel (oriented end-on) to the blast wind.

Blast waves cause injury because of their rapid external loading on the body and organs.[11] Air-containing organs (e.g., middle ear, lung, and GI tract) are the most sensitive to changes in air pressure and are the most susceptible to distortion and stress.[10,11] The more rapid the blast loading increases, the less time internal pressures have to equilibrate, and the tissue distortion and injury in air containing organs will be greater. Thus, primary blast injury is seen almost exclusively in the gas-containing organs.[13] Low levels of tissue stress lead to compromised integrity of vascular beds, leading to local hemorrhage. Higher levels of stress leads to severe tissue disruption and mechanical failure. An overpressure of 5 psi causes tympanic membrane (TM) rupture, 16 psi results in pulmonary blast injury, and 30-42 psi is defined as the lethality threshold pressure.[12,15,16]

Water transmits blast waves more efficiently than air. Compared to air blasts, underwater explosions send out compressive shock waves at much higher speeds (up to 5000 fps).[11,17] After detonations of similar size, underwater blast injuries are more at a given distance than those observed in air blasts.[10] The lethal range of a given size charge underwater blast is about three times that for free-field air blasts.[13,18] As an underwater compressive shock wave reaches the surface, it is reflected as a tension wave. This interacts with the compressive wave at different times resulting in a canceling or "cutoff" effect that is greatest closer to the surface. Therefore, the greatest impulse loading occurs towards the deepest parts of a floating structure. Gastrointestinal injuries are much more likely from blast waves in water than from those propagated in air.[13,14,17,19,20] A person in danger of an impending underwater blast should float on the surface instead of treading water. Fortunately, water greatly reduces the effective range of any fragments propelled from the blast.

MECHANISMS AND DETERMINANTS OF INJURY

Mechanisms and determinants of physical injury from blasts are multifactorial and include: type and size of the blast, distance from the explosion, effects of environmental pressure changes, conditions caused by blast pressures and blast winds, and the environment in which the blast occurs. The organs most often injured by blasts are the ears, skin, lungs, bowels, cardiovascular and central nervous system (CNS).[21] The most common serious effects include respiratory injuries, gastrointestinal damage, and the introduction of air emboli into the circulation system.[19]

Primary blast injury is the direct, cussive effect of the pressure wave on the victim.[13] The true incidence of primary blast injury is not known. Primary blast injury is more likely to occur

when the detonation has occurred in a closed space.[3,17] Categories of primary blast injury are shown in **Table 1**. The amplitude of the peak overpressure and the rate of pressure rise and its total duration determine the biological effects of primary blast injury.[3] Because the pressure amplitude generated by explosions is inversely related to the cubic distance from the focus of detonation,[13] the presence of severe pulmonary or intestinal primary blast injury is evidence of the victim's relative proximity to the explosion.[3] Pulmonary damage should be expected in 50% of victims exposed to blast pressure of ≥ 70 psi.[14] Exposures to pressure levels of ≥ 80 psi are lethal in more than 50% of cases.[22]

Table 1: Categories of Primary Blast Injury

From Sharpnack DD, Johnson AJ, Phillips YY: The pathology of blast injury, in Bellamy RF, Zajtchuk R (editors): Conventional Warfare: Ballistic, Blast, and Burn Injuries. Textbook of Military Medicine Series, Office of the Surgeon General at TMM Publications, Washington, D.C., 1991.

Respiratory System
Pulmonary Hemorrhage
Alveolovenous fistula (air-embolism production)
Airway epithelial damage

Circulatory System
Cardiac contusion
Myocardial ischemic change (air-embolism production)

Digestive System
Gastrointestinal hemorrhage
Gastrointestinal perforation
Retroperitoneal hemorrhage
Ruptured spleen or liver

Eye and Orbit
Retinal air embolism
Orbital fracture

Auditory System
Tympanic membrane rupture
Ossicular fractures
Cochlear damage

Open air blasts may hurl objects through the air that can cause penetrating or nonpenetrating *secondary blast injury*.[3,10] Fragments propelled by conventional blasts travel in the air far beyond the distance that the blast will cause injury.[11] Most of the severe injuries among civilian victims of terrorist bombings are attributed to secondary blast effects as shrapnel wounds.[3] Wounds inflicted by these fragments do not differ from classic ballistic wounds caused by bullets or fragments from conventional explosive munitions.[17] *Tertiary blast injury* results from blunt trauma that occurs when the victim is lifted and thrown against a structure by the blast wave or blast winds.

Flash burns result from the intense heat of explosions,[8] but are usually superficial because of the short duration. Other *thermal injuries* from radiation, hot gases, or fires are considered miscellaneous blast effects.[13] Burns sustained by victims of explosions in closed spaces are more severe and affect a larger percentage of total body surface area.[3] The term *combined blast injury* is used when primary blast injury occurs with secondary or tertiary blast injuries or with burns or radiation.[19] Smoke and fumes from fires resulting from the blast may produce inhalation or toxic injuries. Additionally, crush injury from collapse of a structure is another indirect effect of bombings.

The concepts of spallation, implosion, and inertial effects help define the effects that the force of the blast wave has on tissue in the body.[8,13,21] Spallation is the tendency for a boundary between two different density media to disrupt when a compression wave in the denser medium is reflected at the interface. Implosion is the forceful compression of a gas bubble by a shock wave in liquid that results in the pressure in the bubble rising much higher than the shock pressure. As the pressure wave passes, the bubble re-expands explosively disrupting local tissue. Two adjacent objects of different densities being acted upon by the same force can cause inertial effects. The lighter object will be accelerated more than the heavier one, and great stress will take place at the boundary between the two.

The organ most sensitive to primary blast effect is the ear.[21,23] The blast pressure wave is amplified along the auditory conductive pathway increasing the ear's sensitivity to levels of blast overpressure that might not be sufficient to cause primary blast injury in other organs.[19] Auditory damage from blasts includes rupture of the tympanic membrane, dislocations or fractures of the ossicular chain, and damage to the organ of Corti within the cochlea. Perforations usually involve less than one-third of the tympanic membrane.[13] In a series of 147 patients who sustained blast injury, the distribution of perforations were the inferior part of the eardrum in 48% of the cases, the superior part in 23%, central kidney-shaped in 13%, combined superior and inferior in 15%, and marginal in 6.6%.[24] Hemorrhage in the tympanic membrane is

often around the periphery of the pars tensa or immediately below the anterior or posterior malleal folds.[25] Most perforations heal spontaneously without sequelae; cholesteatoma is a reported complication that is confined to patients in whom the membrane did not heal within 10 months and in whom tympanoplasty was not subsequently performed.[24] Ossicular damage includes medial displacement of the malleal handle with disruption of the incudomalleal joint, and incudostapedial joint separation with and without stapes fracture.[19]

Transient sensoneural loss usually resolves within the first few hours after a blast, but 30% of bombing victims may have permanent loss.[17,26] Tinnitus tends to parallel sensoneural hearing loss and usually resolves as the hearing loss does. One-year audiologic monitoring of 83 survivors of the 1995 Oklahoma City Federal Building bombing showed that 76% still reported tinnitus, 58% distorted hearing, and 57% otalgia.[7]

Injury to the lung is the greatest cause of morbidity and mortality.[13] Primary blast injury to the chest results in damage to the alveolar parenchyma.[27] The blast wave may also produce tears in the visceral pleura, as well as the formation of bullae and alveolar-venous fistulae. In experimental animal studies, more than 50% of the deaths from primary blast injury occurred with the first 30 minutes; autopsies showed many air emboli.[19] The most obvious and consistent sign of primary blast injury to the lungs is hemorrhage, which can manifest externally by froth or blood in the oral cavity or surrounding the nose or lips.[19] The hemorrhage may be pleural or subpleural, multifocal or diffuse within the parenchyma, or surrounding the airways and vascular structures within the parenchyma. Intra-alveolar hemorrhage plus atelectasis probably results in a decrease in functional residual capacity, a lowering of the ventilation:perfusion ratio, and an increase in right-to-left shunting of blood through non-ventilated alveoli.[27]

The characteristic clinical pattern of primary pulmonary blast injury is rapid respiratory deterioration with progressive hypoxia, leading to a need for mechanical ventilatory support with high FIO_2.[3] Widespread pulmonary edema does not usually occur as a result of primary blast injury. Only a small number of terrorist bombing survivors have primary pulmonary blast injury that results in respiratory failure and high in-hospital mortality.[23] This is because persons close enough to sustain such injuries from air blasts are likely killed immediately by the secondary and tertiary effects or by massive air embolism from primary blast injury.[4,14]

Other lung injuries include pneumothorax, hemothorax, traumatic lung cysts, interstitial emphysema, pneumomediastinum, and subcutaneous emphysema. Chest wall damage, including rib fractures, is not usually seen in the absence of other pulmonary trauma.[13] Air emboli, which can result from traumatic alveolovenous fistulae, are responsible for most of the

early mortality from primary blast injury.[13] The more severe the pulmonary hemorrhage, the greater the likelihood of significant embolism.[19] Pulmonary symptoms in survivors of major blasts include: hemoptysis (55%), dyspnea (38%), and chest pain (22%). Clinical findings include: parenchymal infiltrates (84%), crackles (40%), hemothorax (27%), pneumomediastinum (27%), and pneumothorax (4%).[17] In one series of 12 patients with blast injury to the chest, four (33%) presented with cyanosis and ten had severe associated injuries, including nine who required laparotomy.[18]

Blast injury has similar effects in the abdominal cavity as in the thorax: (a) the blast wave strikes and displaces the body wall, causing tissue distortion within leading to their stress and failure; (b) no visible external injury on the body wall; (c) gas-containing organs are most vulnerable; and (d) the most common lesions are hemorrhage and tearing of tissue.[19] Gastrointestinal hemorrhage is most common in the lower small intestines or cecum where gas commonly accumulates. The most common site of intestinal perforation is at the ileocecal junction. Perforation may occur immediately at the time of the blast injury or may delayed up to days later. Retroperitoneal hemorrhage and damage to solid organs are less common, and more likely are the result of secondary or tertiary blast injuries.[17] In a combined series of 43 hospitalized patients from an underwater blast, 67% had perforated bowel, 2% with isolated bowel hematoma, and 2% with solid visceral injury.[18,20]

Circulatory system injury may be a direct result of the blast wave itself; however, indirect effects such as air emboli from pulmonary injury can occlude coronary arteries and cause cardiac arrest. Hemorrhage (myocardial contusion) is the most common blast-related lesion found in the heart and occurs most often on the diaphragmatic surface.[19] Myocardial lacerations have also been documented. Coronary artery air embolism is thought to be a major cause of death in blast casualties.

Traumatic amputations are a hallmark of grave prognosis among blast victims. Most cases are caused directly by shock waves, but some result from large secondary missile fragments.[3] Because most traumatic amputations reflect a high energy transfer, such patients have a high likelihood of dying from severe associated injuries.[2]

Fragments cause most of the injuries that result from small terrorist bombings.[23] Soft tissue injuries (lacerations, abrasions, and contusions) predominate among survivors of bombings.[4,6,28] Bombs characteristically cause multiple wounds with associated gross soiling.[29] The wounds vary in size from minute punctures to huge lacerations often impregnated with foreign bodies. Besides bomb fragments and flechettes, foreign bodies found in wounds may include clothing, stones, glass, splintered wood, and plaster. Fine particles of dust may be

9

embedded within the skin producing a discoloration called "dust tattooing."[14] The head and neck are the most frequently injured regions of the body, followed by the extremities.[28] Clothing and footwear provide a protective effect against soft tissue injuries to the covered areas of the body.[28]

Blast injuries to the eyes usually occur as a result of secondary blast effects; most frequently glass fragments.[30,31] Eye injuries from terrorist blasts can be extensive and involve the globe, the ocular adnexa, and the orbit. In one study of 55 patients with ocular injuries after a terrorist bombing, lid/brow lacerations occurred in 36%, penetrating globe injures in 22%, orbital fractures in 11%, retinal detachment in 9%, and retained foreign body in 4%.[30]

EPIDEMIOLOGY OF BOMBING INJURIES

Terrorist blast-injury data are limited by the chaotic nature of an unexpected mass-casualty situation descending upon a civilian medical system. Critical analysis of the patterns of injury and mortality from this type of disaster help bring understanding of the natural history and pathophysiology of this form of terrorism.[32] A collective profile of injury patterns among 3949 terrorist bombing victims is shown in **Figure 2**.
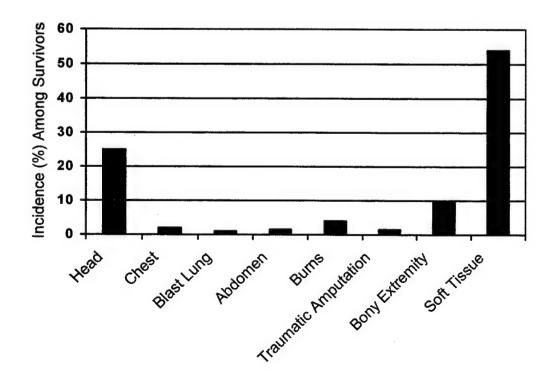
Figure 2: Injury Patterns Among Terrorist Bombing Victims[5,6,32]

The mortality and morbidity of blast injuries largely depends on which organs have been affected.[23] Trauma to the central nervous system is highly lethal. The incidence of head injuries from terrorist bombings ranges from 10.8 - 63%.[5,14,21,28,33-36] About 1-5% of civilian bombing victims will die at the scene from head injuries. Most casualties treated in Emergency Rooms will have minor injuries consisting of lacerations, abrasions, and contusions.[6] About 21-50% will require admission for observation, with a small number of these (1-15%) having significant thoracic or abdominal injuries. Truncal injuries are few (2.4% of admissions), but are more likely to result in death among hospitalized patients. Hospital mortality for bombing victims with chest trauma is 15%: with abdominal injuries it is 19%.[23]

Mortality rates for bombing can be deceptive. The casualties should be broken down into immediate on-scene mortality, number of critically injured survivors (ISS >15), and overall mortality in hospitalized survivors.[32] Basing the overall mortality rates on total casualties will cause an artificial dilution since many bombing incidents have many patients with minor injuries that require minimal treatment without hospitalization. The on-scene mortality of terrorist bombings ranged from less than 2% in Northern Ireland to almost 70% for the Beirut marine barracks attack in 1983.

Higher fatality rates can be anticipated when one or more of the following occurs: structural collapse of buildings, large TNT-equivalent explosion, and explosions in confined spaces (e.g., vehicles, building interiors).[3,5,32] The 1980 bombing of the central railway station in Bologna, Italy; the 1983 bombing of the U.S. Marine compound in Beirut, Lebanon (69% fatality rate); and the 1995 bombing of the Murrah Federal Building in Oklahoma City, Oklahoma (22% fatality rate) produced high fatality rates from building collapse.[5,32,33,35] In one report of 55 casualties in a civilian bus bombing, 11 suffered lung injuries and 4 had gastrointestinal trauma.[15]

In a review of 200 bombing incidents worldwide involving 3357 casualties, Frykberg and Tepas found that the immediate on-scene fatality rate was 13%.[4] The mean number of casualties per bombing was 15. Of the 87% who were immediate survivors, 30% were hospitalized, and 18.7% were critically injured. The mortality rate for immediate survivors was 2.3% and occurred in those deemed critically injured; therefore, the "critical mortality rate" was 12.4%. Head injury was the most common contributor to early and late deaths (71% and 52%, respectively). Surgical procedures performed on 812 survivors of terrorist bombings included 53 (67%) for soft tissue injuries, 142 (17.5%) for orthopedic injuries, 45 (5.5%) for abdominal injuries, 17 (2%) for head injuries, and 65 (8%) for miscellaneous injuries (chest, ear, vascular, neck, spinal cord, peripheral nerve).[4]

Burn fatalities are not high among bombing victims: the brief flash of explosions limits thermal injuries to superficial burns on exposed body areas. [4] High explosive forces, explosions in confined spaces, or secondary fires from the blast may result in more severe burn injuries and mortality.[4,37,38] Smoke inhalation injuries from bombings are unusual, except in enclosed spaces. After the New York City World Trade Center bombing in 1993, 485 out of 546 (88%) of victims treated at surrounding hospitals over a 24-hour period were given a primary diagnosis of smoke inhalation.[39] The bomb knocked out the building's power, disabling the smoke control system, and trapping people within the 198 elevators for long periods while smoke filled the lift shafts.

Primary blast injury is quite common in terrorist bombings, but most of these victims are on-scene fatalities due to the associated injuries.[10] Primary blast injury to the lung was noted in only 0.6% of 2934 immediate survivors of bombings.[4] Autopsies of 495 casualties who died in 5600 separate explosions over a 12-year period showed that 66% had brain injuries, 51% had skull fractures, 47% had classic autopsy findings of blast lung, 45% had tympanic membrane rupture, and 34% had liver lacerations.[10]

Explosions in an enclosed space result in more severe primary blast injury. Of 104 passengers on two civilian buses that were bombed in Israel, 7 were killed immediately, 51 were admitted to the hospital, 16 had pulmonary blast injury, and 7 complained of significant abdominal pain, with 4 having intestinal perforations.[15,23] All of the severely injured also had perforated tympanic membranes. At least 42 of the 80 Americans injured in the 1986 LaBelle Disco terrorist bombing in Berlin had ruptured tympanic membranes.[40]

The outcomes of victims of terrorist bombing can be categorized as immediate deaths, immediate survivors treated as outpatients, immediate survivors hospitalized, and deaths among immediate survivors hospitalized. **Figure 3** depicts the victims' outcomes in several terrorist bombings. Several terrorist bombings are then briefly described to illustrate the diverse nature of such incidents.
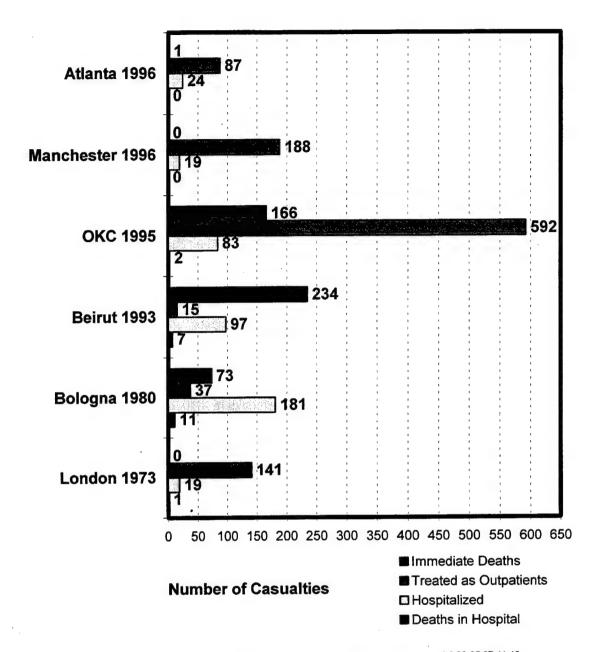
Figure 3: Victim Outcomes in Terrorist Bombing Incidents[4-6,32-35,37,41,42]

Bologna, Italy, 1980

On August 2, 1980, a suitcase bomb exploded in the waiting room of the central railway station in Bologna, Italy.[33] There were 73 fatalities at the scene, the majority due to crush injury when the building collapsed. Of the 218 wounded who initially survived, 181 (83%) were admitted to hospitals. Seventeen (8%) had severe or critical injuries. Among the immediate survivors, there were 43 head injuries including 10 skull fractures, 6 with concussions, and 27

with contusions; 15 ear injuries; 7 eye injuries; 4 pneumothoraces; 4 pulmonary contusions; 7 abdominal injuries; 55 fractures; 28 burns; and 72 other soft tissue wounds.

Forty-one (23%) of the hospitalized patients required surgery for soft-tissue injuries, 22 (12%) for fractures, 6 (3%) for abdominal injuries, 6 (3%) for neurosurgical injuries, and 1 (0.6%) for thoracotomy. Forty-five (21%) of these required general anesthetic. More than 100 units of blood were used in the first 24 hours for these bombing victims.[33]

Beirut, Lebanon, 1983

On October 23, 1983, a yellow Mercedes truck loaded with an estimated 6 to 12 tons of TNT was driven into the ground floor lobby of the U.S. Marines Battalion Landing Team Headquarters building in Beirut, Lebanon, and detonated.[32,35,37] This four-story building that also served as billets for the troops and was occupied by more than 350 men collapsed "like a house of cards."[37] There were 234 men (68%) immediately killed. Three field triage stations were established at the scene.[32,35] Eight casualties were evacuated to local Lebanese hospitals, 72 were evacuated by helicopter to USS *Iwo Jima*, which had a mobile surgical team aboard; 61 of these were evacuated within two hours of the bombing. Six hours after the bombing, another 24 casualties were evacuated by British Royal Air Force, and U.S. Navy and Air Force air ambulances to Germany, and subsequently to the United States. Fifteen survivors with minor injuries were treated and did not require evacuation.

Among the immediate survivors, there were 37 head injuries, including 28 with concussions and 13 with skull fractures.[35] Seven (25%) of those suffering concussions developed post-concussion syndrome.[35] Head injury was the most common (71%) specific cause of death in immediate fatalities in Beirut. Despite intensive medical care, there were 7 deaths (7.3%) among the survivors who were evacuated (three head injuries, one chest injury, one with head and chest injuries, and two major burns).[37] Two of these deaths occurred within 9 hours of the bombing: one with primary pulmonary blast injury, and one with crush injuries to the head and chest.[32] Among the delayed fatalities, 6 of 7 (86%) were extricated between five and nine hours after the blast. Burn victims had the highest specific hospital mortality rate, 40% (two of five). Most of the evacuated survivors suffered multiple injuries. Soft tissue and orthopedic procedures were the most common surgeries performed. Six patients had craniotomies.

Oklahoma City, Oklahoma, 1995

On April 19, 1995, a truck bomb consisting of more than 4000 pounds of ammonia nitrate was detonated outside the Alfred P. Murrah Federal Building in Oklahoma City (OKC).[6] There were 163 immediate deaths and three dead on arrival at local hospitals. There were 388 victims treated in emergency departments the day of the bombing.[6] Eight-three (21%) immediate survivors were hospitalized , and 2 (2.4%) of these subsequently died.[5] One of these died on the second hospital day due to a head injury. The other died three weeks later of multiple organ failure and sepsis, the usual cause of late bombing victim deaths (days to weeks after the blast). The medical examiner did not clearly distinguish deaths caused by primary blast injuries from those caused by building collapse in those victims who were on-scene fatalities; the most common cause of death reported was simply "multiple injuries."[5]

The majority of the survivors sustained minor injuries caused by flying or falling glass and debris. The most frequent type injury requiring emergency department treatment was soft tissue trauma in 71% followed by fractures in 12%.[6] Blast injury to the eye was documented in 8% of the victims and was much more common in victims who were in the building versus other locations.[30] Abdominal trauma was not common in the OKC bombing, with only one splenic injury and two renal contusions reported.[6] The most frequent medications administered in the emergency departments were tetanus immunizations, analgesics and antibiotics.

The truck bomb detonated in front of the Murrah Building resulted in several secondary fires. Ten victims suffered thermal burns: one fatality occurred in the parking lot, and seven were hospitalized with up to 70% burns.[1,5] Among the nine survivors, the most common area burned was the face and neck (67%); no patient had full-thickness burns involving more than 10% of body surface area.

Manchester, England, 1996

On June 15, 1996, the largest bomb ever detonated by the Irish Republic Army in Great Britian exploded in the center of Manchester, England.[41] There were no fatalities, but 208 victims were treated in the nearest five hospital emergency departments; most (62%) for injuries from flying glass. This placed a large burden on the hospitals' radiology departments, since 50% of the casualties required radiologic studies. There were 41 (20%) who suffered blunt trauma. Admission was required for 18 (9%) and eight underwent surgery. An unusually high number of patients (36; 17%) presented for medical problems (e.g., angina, asthma attack, syncope) or emotional distress related to the bombing.

15

Atlanta, Georgia, 1996

In July 1996, a pipe bomb in a knapsack filled with nails and screws exploded in Centennial Park during the Atlanta Olympics.[42] The bomb detonated in front of a concert stage 75-100 yards away from attendees who were being evacuated. There were 111 patients treated in local hospitals. One additional patient suffered a fatal heart attack while running to the scene of the bombing. Hospital admission was required for 24 (22%). Virtually all of the patients were treated for wounds related to fragments from the pipe bomb, or the nails and screws.

The impact of a terrorist bombing is not limited to the dead and injured.[43] The psychological impact on the surrounding community may result in behavioral consequences, such as changes in smoking and drinking habits, Post-Traumatic Stress Disorder (PTSD), and intrusive thoughts.

MANAGEMENT OF CONVENTIONAL BOMBING VICTIMS

PRE-HOSPITAL

Extrication and life support are the first priorities.[8] Bombing victims should be evaluated according to normal basic trauma triage standards.[13,17] After terrorist bombings, rapid identification of life-threatening injuries and evacuation of severely injured victims to appropriate medical facilities are paramount. Severely injured bombing victims will have higher survival and lower morbidity if transported to a trauma center, not simply to the closest hospital.[35] Patients with asphyxia, suspected simple or tension pneumothorax, cyanosis or extreme dyspnea, upper-airway compromise, or hypotension should be triaged as immediate. Only a minority of survivors of bombings have severe life-threatening injuries.[6,28,32-34,36,44,45] Common pre-hospital interventions required include spinal immobilization, wound dressings, and intravenous fluids.[6]

Rescue and recovery of injured victims after a bombing may be hampered by further terrorist activity. After the 1983 Beirut Marine Headquarters bombing, rescue efforts at the scene were disrupted by sniper fire throughout the day.[37] A second bomb specifically designed to maim or kill personnel rushing to help those wounded in a first explosion is a well-recognized terrorist tactic.[8,46] In 1996, a second terrorist bomb specifically targeting medical staff complicated the bombing of the Thiepval British Army Barracks.[46] Additionally, rescuers must be trained and prepared for terrorist bombings that use nuclear, biological, or chemical agents

16

as secondary weapons of mass destruction. The presence of collapsed buildings with trapped victims prolongs the recovery phase and creates more severely injured patients.[6,47]

Primary blast injury is likely to be only part of the presentation of a multiply-injured bombing victim.[14] The pre-hospital care of bombing victims usually centers on secondary blast injuries such as fractures, amputations, penetrating wounds, and burns. However, pre-hospital providers should be particularly alert for subtle signs of primary blast injury, especially for pulmonary injuries such as tension pneumothorax. Hypovolemic shock may occur as a result of hemorrhage from wounds or by blood loss from gastrointestinal injuries.

All patients with other than isolated minor soft-tissue injuries should receive supplemental oxygen and monitored with pulse-oximetry. Patients with extreme respiratory distress should be intubated endotracheally to handle massive hemoptysis and to provide ventilatory support. With mechanical ventilation comes the increased risk of iatragenic tension pneumothorax in blast victims. Patients may need prophylactic tube thoracostomies with Heimlich valves before aeromedical evacuation.

Table 2 outlines information pre-hospital medical personnel should attempt to obtain about the blast as soon as possible after the casualties are stabilized and circumstances permit.[17]

Table 2: History to Obtain at the Scene of a Bombing Casualty[17]

◆ What type of ordnance was used and how large was the explosion?

◆ Where was the casualty located with respect to the blast?

◆ Did the blast occur inside an enclosed space such as a room or vehicle?

◆ What was the casualty's activity after exposure?

◆ Were there fires or fumes that might lead to an inhalation injury?

◆ What was the orientation of the casualty's head and body to the blast?

Victims of explosions should minimize post-blast physical activities and be kept sedentary to avoid exacerbation of the severity of primary blast injury.[13] Those who are experiencing any respiratory distress should be carried from the incident scene on a litter. Manifestations of primary blast injury involving the gastrointestinal, respiratory and circulatory systems are more dangerous if the victim is transported by air; therefore, even short helicopter flights should be avoided.[17]

Patients with injured lungs are at risk for air embolism, which is the single most important cause of death from primary blast injury.[10] If one lung is more severely affected than the other is, the damaged lung should be in the dependent position during transportation.[17] The alveolar pressures throughout the dependent lung will be lower than vascular pressures decreasing the risk of air embolism, but at the risk of worse gas exchange. The position of the patient's body may affect the site of embolism travel. Therefore, if the lungs are equally affected or the left lung is more severely injured, the patient should be placed in the left-lateral decubitus and Trendelenburg positions. High-flow oxygen should be administered to support gas exchange in the lungs and help the tissue absorption of the emboli by decreasing the predominance of nitrogen gas.

EMERGENCY DEPARTMENT

Blast victims should be triaged upon arrival to the ED by an experienced trauma surgeon or emergency physician. Unfortunately, the extent of blast injury cannot be reliably assessed by the typical rapid triage examination.[48] This mandates an atypically high over-triage rate,[4,32] which may affect patient flow and use of hospital resources.[48]

The emergency department care should be centered on stabilization measures in accordance with Advanced Trauma Life Support principles.[49] A thorough evaluation for signs of primary blast injuries should also be conducted; especially when there is a history of a powerful explosion. Physical examination findings such as ruptured tympanic membranes, hypopharyngeal contusions, hemoptysis in the absence of signs of external chest trauma, or subcutaneous emphysema indicate primary blast injuries. In fact, a ruptured eardrum is almost always present when the blast pressure is high enough to cause serious injury to the lung or gut.[13] Petechiae or ecchymoses in the hypopharynx or larynx may be associated with significant pulmonary injury; their absence speaks against exposure to high blast pressure.[13] Signs of primary blast injury to the lung also include dyspnea, cough, restlessness, tachypnea, tachycardia, cyanosis, or an inability to carry on a conversation.[17,21] Immediate tube
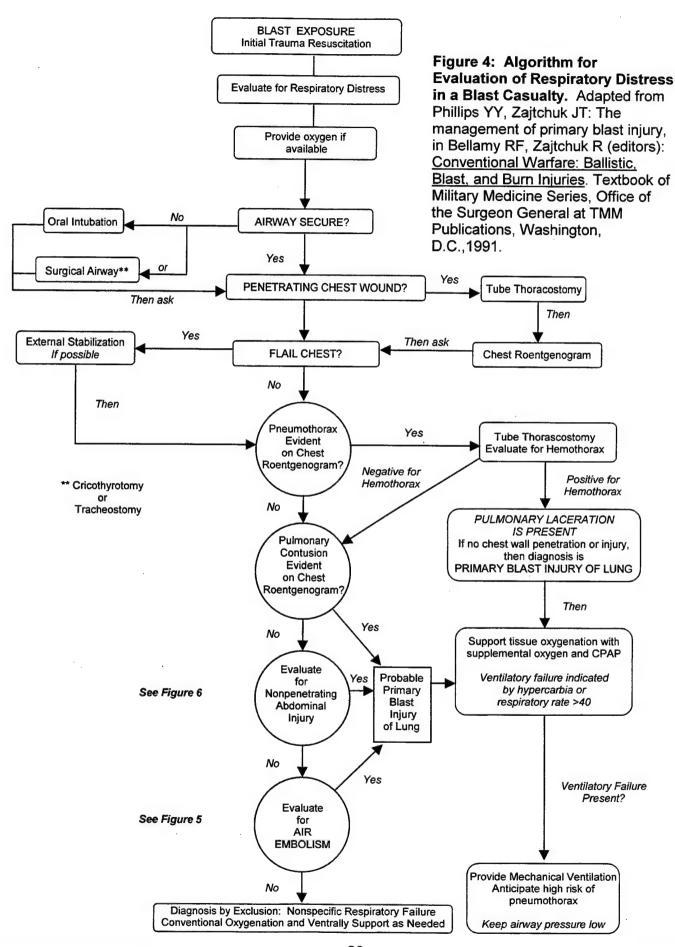
18

thoracostomy is indicated for pneumothorax, tension pneumothorax, or hemothorax. A chest radiograph should be routine in all blast victims (except those with isolated minor soft tissue injuries of the extremities from air blasts) since it may reveal pulmonary lesions not evident clinically.[33]

Radiographic findings in blast injury to the chest are non-specific, but include pneumothorax, hemothorax, pneumomediastinum, atelectasis, alveolar infiltrates, and interstitial edema.[18] Computed tomography (CT) of the chest is the most accurate technique for evaluating the lung parenchyma and pleural space and is used to quantitate the extent of the injury based upon the amount of parenchyma involved.[17] Chest CT scans may also reveal underlying parenchymal injury or pneumothorax when extensive subcutaneous emphysema, parenchymal hemorrhage, or interstitial emphysema is present on plain films.

The main objective of treatment of blast lung is to restore and maintain arterial blood gases near normal until the lungs have recovered.[27] Patients with more than 28% of their lungs involved with hemorrhage usually require mechanical ventilation; however, this increases the incidence of both air emboli and thoracic barotrauma. Continuous positive pressure ventilation (CPPV) should not be withheld if the clinical situation deteriorates, but patients should be continuously re-evaluated for the development of a tension pneumothorax.[17,25] Airway pressures should be kept as low as possible. To promote good bronchial hygiene by using bronchodilators, removing of blood and secretions from the tracheobronchial tree by frequent suctioning, and frequently changing the patients body position.[17,27]

Arterial blood gases should be obtained for seriously injured patients and those with respiratory distress. Generally, victims with uncomplicated primary blast injury have a normal or low pCO_2 (<40 mm Hg). The presence of hypercarbia (pCO_2 > 40 mm Hg) suggests something other than primary blast injury may be limiting ventilation, such as flail chest, muscle weakness from chemical agent exposure, metabolic derangements, airway compromise, impairment of central ventilatory drive, air embolism, excessive narcotic administration, or diaphragm rupture.

An electrocardiogram should be obtained for seriously injured patients including any with hypotension, respiratory or CNS symptoms. Ischemic changes may be due to air emboli to the coronary arteries, or hypoxia secondary to respiratory failure.[21] **Figure 4** is an algorithm for the initial trauma resuscitation and evaluation and treatment of a blast casualty with respiratory distress.[17]

Figure 4: Algorithm for Evaluation of Respiratory Distress in a Blast Casualty. Adapted from Phillips YY, Zajtchuk JT: The management of primary blast injury, in Bellamy RF, Zajtchuk R (editors): <u>Conventional Warfare: Ballistic, Blast, and Burn Injuries</u>. Textbook of Military Medicine Series, Office of the Surgeon General at TMM Publications, Washington, D.C.,1991.

Hypotension is a frequent finding in severe blast injury and may be the result of severe acute blood loss, myocardial injury, vasovagal reflexes, or a combination of these.[20,21] Central venous monitoring may help guide fluid replacement therapy. Hypotensive patients should receive sufficient fluid resuscitation to bring their blood pressure back within normal limits, but those with pulmonary injuries will have an increased risk of pulmonary edema when they receive excessive volume replacement.[17] Blood or a colloid solution is preferred over crystalloid solutions in these patients. Blast casualties with ruptured tympanic membranes or any signs of primary blast injuries, abdominal pain, altered sensorium, or injuries other than minor soft tissue trauma should have a chest radiograph regardless of whether they have any pulmonary symptoms. In one study of 137 bombing patients with isolated eardrum rupture, Leibovici et al predicted that up to 3% may demonstrate delayed pulmonary barotrauma.[50] In a report of 27 survivors of an underwater detonation, 19 were subsequently found to have significant pulmonary compromise without initially presenting with overt respiratory distress.[17] Likewise, abdominal radiographs are indicated in all but the most mildly injured patients.

Air embolism from patients with injured lungs may cause occlusions in any organ and pose the most immediate threat to life. The risk of an air embolus is increased with the use of mechanical ventilation and usually occurs within 2 hours of the blast, but has been reported 60 hours after injury.[17] Retinal artery air emboli may be directly seen on ophthalmoscopic examination as streaming bubbles or pale silvery sections of the vessels or indirectly indicated by retinal pallor. Emboli involving the cerebral or coronary circulation are the most serious and may lead to strokes or myocardial infarctions. Blast victims should be evaluated for headaches, seizures, mental status changes, transient blindness, tongue blanching, vestibular disturbances, focal neurological deficits; and indicators of cardiac distress such as chest pain, hypotension, dysrhythmias, and ischemic changes on electrocardiograms. (**Figure 5**) Direct head trauma from secondary or tertiary blast injury is more likely to be the etiology of neurological signs and symptoms.[13] Early head CT scans and neurosurgical consultation are mandatory for patients with penetrating cranial injuries, altered sensorium, or other neurological findings.
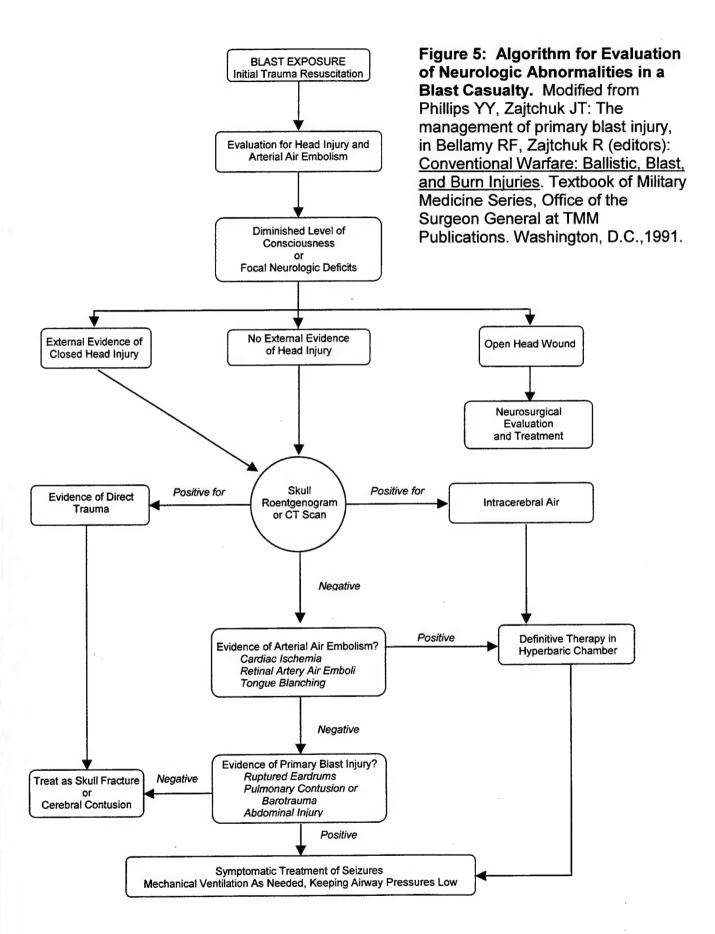
The treatment of choice for life-threatening air emboli is hyperoxygenation in a hyperbaric chamber. Treatment should be started as soon as possible after the blast exposure.[8,13,17] Hyperbaric treatment has been shown to reduce mortality in animal experiments but studies are lacking in human survivors of blasts.

Blast victims with abdominal injuries who survive the first few hours or days may succumb to complications, especially from delayed perforation. Although gastrointestinal injuries may be initially overshadowed by more immediately life-threatening pulmonary or soft-

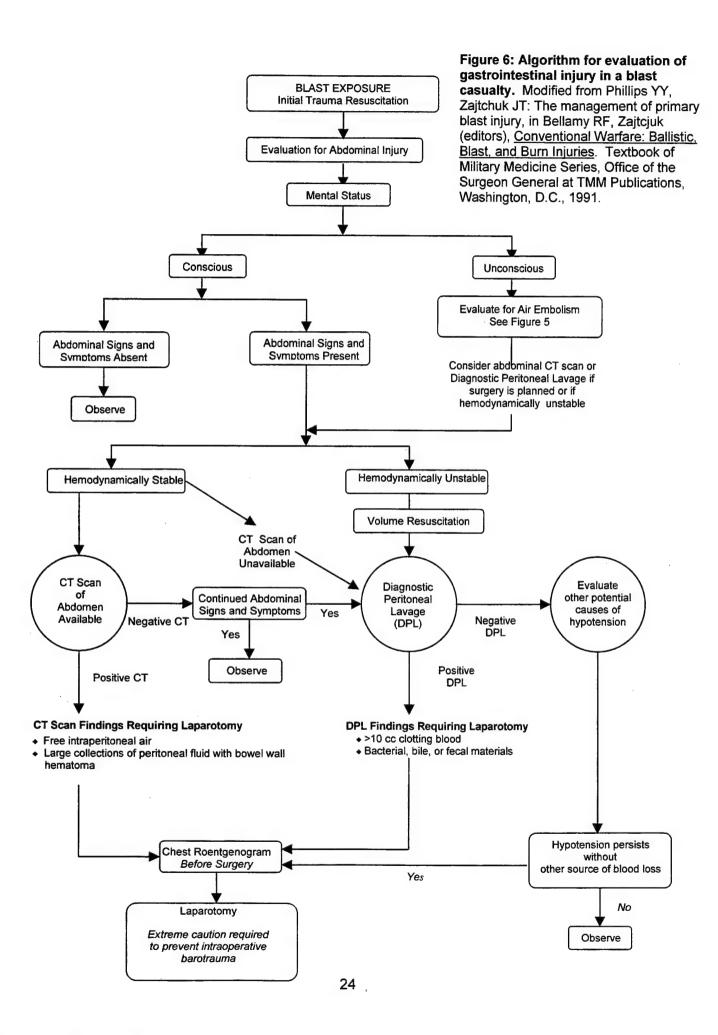tissue injuries, many will require laparotomy for definitive treatment. Symptoms of abdominal injury include abdominal pain, nausea, vomiting, hematemesis, rectal bleeding, orthostasis or syncope, testicular pain, and tenesmus. Findings on physical examination include decreased or absent bowel sounds, gross blood on rectal examination, hypotension, involuntary guarding, and peritoneal signs such as rebound tenderness. Unequivocal signs of peritoneal irritation require prompt surgical treatment.[17] Abdominal radiographs may show free intraperitoneal air.[13] An algorithm for evaluating bombing victims for gastrointestinal injury is shown in **Figure 6**. Patients with penetrating abdominal injuries or those with suspected bowel rupture should be given antibiotics as soon as possible to cover for anaerobes, Gram-negative aerobes and enterococci.

Patients with multi-system injuries, unconscious patients, and those with any signs of abdominal injury can undergo a rapid screening ultrasound of the abdomen in the emergency room looking for free peritoneal fluid. Hemodynamically unstable patients should have a prompt diagnostic peritoneal lavage. Plain abdominal films may not reveal extraluminal air from bowel perforations; therefore, hemodynamically stable patients with signs or symptoms of abdominal trauma should undergo abdominal CT scanning with oral and intravenous contrast. The abdominal CT study should be extended to include the lower chest to evaluate for lung injuries that may not be apparent on chest radiographs.

Symptoms of auditory injury include hearing loss, otalgia, tinnitus, loudness sensitivity, and vertigo. Hearing in both ears should be grossly tested at the bedside. Patients with vertigo should have a complete neurologic examination for signs of closed head injury. All bombing victims should have a thorough otoscopic examination including those who are unconscious. The location and size of perforations should be noted. The external auditory canal and middle ear space should be examined for the presence of foreign material. The presence of clear otorrhea may be due to injuries to the inner ear, such as perilymphatic fistulae in the oval window, dislocated stapes, ruptures of the saccule, urtricle, and basilar membrane;[17] or may be CSF from an middle fossa basilar skull fracture. All patients with signs or symptoms of auditory injury should have complete audiology and otolaryngology evaluations as soon as their clinical conditions permit. Patients with auditory injuries should be followed for one year to document change across time and to provide supportive rehabilitation.[8]

Figure 5: Algorithm for Evaluation of Neurologic Abnormalities in a Blast Casualty. Modified from Phillips YY, Zajtchuk JT: The management of primary blast injury, in Bellamy RF, Zajtchuk R (editors): Conventional Warfare: Ballistic, Blast, and Burn Injuries. Textbook of Military Medicine Series, Office of the Surgeon General at TMM Publications. Washington, D.C.,1991.

Figure 6: Algorithm for evaluation of gastrointestinal injury in a blast casualty. Modified from Phillips YY, Zajtchuk JT: The management of primary blast injury, in Bellamy RF, Zajtcjuk (editors), <u>Conventional Warfare: Ballistic, Blast, and Burn Injuries</u>. Textbook of Military Medicine Series, Office of the Surgeon General at TMM Publications, Washington, D.C., 1991.

24

Topical antibiotics drops are not routinely given to blast injury patients with ruptured tympanic membrane (TM) unless infection is present. As a rule of thumb, one month is required to heal each 10% of the TM rupture. Follow-up examinations of the TM should occur within 2 weeks and then monthly thereafter. Perforations involving less than 1/3 of the TM usually close spontaneously. Ruptures that do not heal spontaneously are treated with paper or Gilfilm patching or by tympanoplasty.[17] Surgical intervention will ultimately be required for 25% of the ruptures.[13] Surgery should be performed 10-12 months after the blast injury to obtain maximal success in closure, and to reduce the risk of chronic infections and inner ear damage.[24]

Patients injured in terrorist bombings frequently have suffered soft-tissue wounds from high-velocity penetrating fragments.[10] All penetrating wounds, no matter how trivial, should undergo local exploration. Wounds should be thoroughly cleansed and irrigated, followed by appropriate debridement.[51] Judicious use of radiographs to look for foreign bodies is also recommended. Delayed primary closure should be the rule.[2,6,8,10,29,36,34] This is important to emphasize to physicians who do not deal with wounds regularly; because, if there are a large number of bombing casualties from a given event, minor wounds will be treated in many other settings besides the ED.

An initial dose of antibiotics should be administered parenterally in the emergency department for all but the most trivial bombing related wounds, followed by a three to five-day course of oral antibiotics. Tetanus immunization is the most frequent medication required in bombing victims,[6,21] and should be administered if indicated. Bombing-related wounds should be considered tetanus-prone wounds.

Open fractures that occur as a result of explosions frequently contain debris and fragments. These should be irrigated copiously in the ED and again in the operating room before and after debridement, followed by external fixation.[2,52] Patients should receive parenteral antibiotics at the earliest opportunity.

The majority of bombing victims will not have life-threatening injuries and will require local wound care. Hospital disaster planners should be prepared to utilize other areas besides the emergency department for this care and keep ED resuscitation areas available for the more seriously injured patients.

HOSPITAL

Positive pressure ventilation or general anesthesia puts casualties with pulmonary primary blast injury at higher risk. Due to high morbidity rate when blast victims receive general

anesthesia, it is recommended that local, regional, or spinal anesthetic be used whenever possible and the duration of surgery kept short.[13,17] Whenever possible, surgery requiring general anesthesia should be postponed 24 to 48 hours.[20] All blast victims undergoing general anesthesia should be monitored for oxygen saturation, end-tidal carbon dioxide, cardiac rhythm, blood pressure, and airway pressure. Patients with suspected pulmonary barotrauma should have chest tubes placed before surgery or should undergo period intraoperative chest radiographs during extended surgical procedures. Equipment for pleural space decompression and tube thoracostomy should be readily available at all times. Hyperbaric oxygen has been recommended in the treatment of pulmonary blast injury and for improved oxygenation and in the treatment of air emboli.[20,53]

The radiographic course of blast lung is consistent except when pneumothorax, atelectasis, or infection occurs.[27] Worsening in the radiographic findings of pulmonary contusion is unusual more than 6 hours post-blast. Routine prophylactic antibiotics should not be given.[8] Most pulmonary hemorrhages and contusions will begin to resolve in 1-2 days, although mechanical ventilation may be required for several days. Suspect a superimposed disease process if the radiographic appearance is more extensive after 48 hours. Antibiotic therapy should be guided by Gram stain and serial sputum cultures. Fiberoptic bronchoscopy is indicated for persistent brisk hemoptysis or refractory lobar atelectasis. This may reveal bronchial fractures and lacerations.

Patients with tenesmus or rectal bleeding should undergo flexible sigmoidoscopy.[17] The surgical treatment of abdominal blast injuries follows that for other blunt abdominal trauma. Gastric decompression with a nasogastric tube is an important adjunct to the surgical care of abdominal injuries and in patients who are receiving mechanical ventilation.

Hospital resource utilization, including manpower needs and patient flow, has been analyzed based upon data from 12 urban terrorist bombing incidents in Israel.[48] The major bottleneck to the flow of critically injured patients was the availability of shock rooms and CT scanners, not operating rooms. Imaging studies required for the critically injured stable patients typically included head, chest or abdominal CT scans, followed by plain radiographs to locate shrapnel and diagnose fractures.

Hospital disaster plans should include protocols to postpone radiographs on non-critical casualties with suspected closed fractures without vascular compromise until injured patients are no longer arriving. Those patients waiting for delayed radiographs can be treated with temporary splints and analgesics.[48]

26

Psychological disturbances may affect up to 50% of bombing survivors, and include hysteria, guilt, PTSD, and behavioral alterations.[28,8] It is important to provide psychologic follow-up opportunities for all patients discharged from the hospital. Most symptoms will improve with time.[54]

SUMMARY

Data from previous conventional terrorist bombings show a consistent pattern of injury and death. The majority of injuries are non-life-threatening minor wounds that can be treated in outpatient settings. The most common primary blast injury among survivors of terrorist bombings is tympanic membrane perforation. Other primary blast injuries are usually immediately lethal, and therefore not frequently seen among survivors. The critical mortality rate is the most accurate indicator of the efficacy of medical care provided to immediate survivors.[4]

There are unique aspects for dealing with victims of conventional terrorist bombs. Disaster plans should include the possibility of conventional terrorist bombings and outline a response plan for the evacuation and treatment of the immediate survivors. Urban hospitals should stockpile sufficient quantities of antibiotics, tetanus immunizations, wound care supplies, and external fixators in preparation for terrorist bombing incidents.

WORD COUNT = 8523

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